AMENDMENTS TO THE CLAIMS

- 17. An isolated estrogen receptor- β comprising the sequence depicted in Figure 4, SEQ ID. NO:2.
- 18. An isolated estrogen receptor- β comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2.
 - 19. A method for identifying hER β -interactive compounds, said method comprising:
- (a) contacting the polypeptide of claim 17 with a labeled ligand in the presence of test compounds, to form test reactions, and in the absence of test compounds, to form control reactions;
- (b) incubating said test and control reactions under appropriate conditions to achieve equilibrium binding of said labeled ligand to hER β ;
- (c) determining the level of binding of said labeled ligand to hER β in said test and control cultures; and
- (d) identifying as a hER β -interactive compound any compound that reduces the binding of said labeled ligand to hER β .
 - 20. A method as defined in claim 19, wherein said ligand is $17-\beta$ estradiol.
- 21. A method as defined in claim 19, wherein said hER β -interactive compound is an agonist.
- 22. A method as defined in claim 19, wherein said hER β -interactive compound is an antagonist.

- 23. An antibody that specifically recognizes hER β .
- 24. A method for identifying hER β -interactive compounds, said method comprising:
- (a) contacting the polypeptide of claim 18, which polypeptide encodes hER β , with a labelled ligand in the presence of test compounds, to form test reactions, and in the absence of test compounds, to form control reactions;
- (b) incubating said test and control reactions under appropriate conditions to achieve equilibrium binding of said labeled ligand to $hER\beta$;
- (c) determining the level of binding of said labeled ligand to hER β in said test and control cultures; and
- (d) identifying as a hER β -interactive compound any compound that reduces the binding of said labeled ligand to hER β .
 - 25. A method as defined in claim 24, wherein said ligand is $17-\beta$ estradiol.
- 26. A method as defined in claim 24, wherein said hER β -interactive compound is an agonist.
- 27. A method as defined in claim 24, wherein said hER β -interactive compound is an antagonist.
- 28. The polypeptide of claim 17, wherein the polypeptide is modified with a label capable of providing a detectable signal.
 - 29. The polypeptide of claim 28, wherein the signal is a radioisotope.
 - 30. The polypeptide of claim 28, wherein the signal is a fluorescent compound.

- 31. The polypeptide of claim 18, wherein the polypeptide is modified with a label capable of providing a detectable signal.
 - 32. The polypeptide of claim 31, wherein the signal is a radioisotope.
 - 33. The polypeptide of claim 31, wherein the signal is a fluorescent compound.
 - 34. The polypeptide of claim 17, wherein the polypeptide is produced in intact cells.
- 35. The polypeptide of claim 17, wherein the polypeptide is produced in cell-free translation systems.
 - 36. The polypeptide of claim 18, wherein the polypeptide is produced in intact cells.
- 37. The polypeptide of claim 18, wherein the polypeptide is produced in cell-free translation systems.
 - 38. The polypeptide of claim 17, wherein the polypeptide is chemically synthesized.
- 39. The polypeptide of claim 17, wherein the polypeptide is produced in a recombinant system.
 - 40. The polypeptide of claim 18, wherein the polypeptide is chemically synthesized.
- 41. The polypeptide of claim 18, wherein the polypeptide is produced in a recombinant system.
- 42. (New) A purified polypeptide comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein when this polypeptide forms the N-terminus of human estrogen receptor β , the estrogen receptor β stimulates estrogen response element (ERE) activity to a greater extent than the truncated estrogen receptor lacking this N-terminal polypeptide sequence.

- 43. (New) A purified polypeptide comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein when this polypeptide forms the N-terminus of human estrogen receptor β , the estrogen receptor β attenuates NF-kB transcription activation while the truncated estrogen receptor lacking this N-terminal polypeptide sequence does not.
- 44. (New) A purified polypeptide comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein when this polypeptide forms the N-terminus of human estrogen receptor β , the estrogen receptor β is 2 to 3 times more active than the truncated estrogen receptor lacking this N-terminal polypeptide sequence in activating the ERE-reporter gene in the presence of estradiol.
- 45. (New) An isolated estrogen receptor- β comprising an N-terminus having amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein the estrogen receptor β stimulates ERE activity to a greater extent than the truncated estrogen receptor lacking this N-terminal polypeptide sequence.
- 46. (New) An isolated estrogen receptor- β comprising an N-terminus having amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein the estrogen receptor β attenuates NF-kB transcription activation while the truncated estrogen receptor lacking this N-terminal polypeptide sequence does not.
- 47. (New) An isolated estrogen receptor- β comprising an N-terminus having amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein the estrogen receptor β is 2 to 3 times more active than the truncated estrogen receptor lacking this N-terminal polypeptide sequence in activating the ERE-reporter gene in the presence of estradiol.